

## RET Protein, Human, Recombinant (aa 658-1114, His & GST)

### General Information

Synonyms:	HSCR1;RET51;CDHR16;RET-ELE1;ret proto-oncogene;MEN2A;PTC;MTC1;CDHF12;MEN2B
Protein Construction:	A DNA sequence encoding the cytoplasmic domain of human RET (P07949-1) (His 658-Ser 1114) was fused with the N-terminal polyhistidine-tagged GST tag at the N-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	Baculovirus Insect Cells
Accession:	P07949-1
Molecular Weight:	76.7 kDa (predicted); 70 kDa (reducing conditions)

### QC Testing

Biological Activity:	The specific activity was determined to be 17 nmol/min/mg using synthetic TRK-C-derived Peptide (R11-VYSTDYRLFNPS) as substrate.
Purity:	> 90 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Supplied as sterile 20 mM Tris, 500 mM NaCl, 10% gly, pH 8.0.

### Preparation and Storage

#### Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

#### Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

#### Shipping:

Proteins are shipped with blue ice.

### Protein Background

RET proto-oncogene, also known as RET, is a cell-surface molecule that transduce signals for cell growth and differentiation. It contains 1 cadherin domain and 1 protein kinase domain. RET proto-oncogene belongs to the protein kinase superfamily, tyr protein kinase family. RET proto-oncogene is involved in numerous cellular mechanisms including cell proliferation, neuronal navigation, cell migration, and cell differentiation upon binding with glial cell derived neurotrophic factor family ligands. It phosphorylates PTK2/FAK1 and regulates both cell death/survival balance and positional information. RET is required for the molecular mechanisms orchestration during intestine organogenesis; involved in the development of enteric nervous system and renal organogenesis

during embryonic life; promotes the formation of Peyer's patch-like structures; modulates cell adhesion via its cleavage; involved in the development of the neural crest. RET proto-oncogene is active in the absence of ligand, triggering apoptosis. RET acts as a dependence receptor; in the presence of the ligand GDNF in somatotrophs (within pituitary), promotes survival and downregulates growth hormone (GH) production, but triggers apoptosis in absence of GDNF. It also regulates nociceptor survival and size; triggers the differentiation of rapidly adapting (RA) mechanoreceptors; mediated several diseases such as neuroendocrine cancers. Defects in RET may cause colorectal cancer, hirschsprung disease type 1, medullary thyroid carcinoma, multiple neoplasia type 2B, susceptibility to pheochromocytoma, multiple neoplasia type 2A, thyroid papillary carcinoma and congenital central hypoventilation syndrome. Cancer ImmunotherapyImmune CheckpointImmunotherapyTargeted Therapy

### Reference

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- Garcia-Lavandeira M, et al. (2012) Craniopharyngiomas express embryonic stem cell markers (SOX2, OCT4, KLF4, and SOX9) as pituitary stem cells but do not coexpress RET/GFRA3 receptors. *J Clin Endocrinol Metab.* 97(1):E80-7.
- Stine ZE, et al. (2011) Steroid hormone modulation of RET through two estrogen responsive enhancers in breast cancer. *Hum Mol Genet.* 20(19):3746-56.
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